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Antibiotic Therapy of Aortic Graft Infection: Treatment and Prevention Recommendations

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Surgical site infection (SSI) after aortic intervention, an uncommon but serious vascular condition, requires patient-specific antibiotic therapy. Effective treatment and prevention requires the vascular surgeon to be cognizant of changing SSI microbiology, advances in antibiotic delivery, and patient characteristics. The majority of aortic graft infections are caused by Gram-positive bacteria, with methicillin-resistant *Staphylococcus aureus* now the prevalent pathogen. Nasal carriage of methicillin-sensitive or methicillin-resistant *S aureus* strains, diabetes mellitus, recent hospitalization, a failed arterial reconstruction, and the presence of a groin incision are important SSI risk factors. Overall, the aortic SSI rate is higher than predicted by the Centers for Disease Control and Prevention's National Nosocomial Infections Surveillance risk category system; ranging from 5% after open or endovascular aortic interventions to as high as 10% to 15% after aortofemoral bypass or uni-aortoiliac grafting with femorofemoral bypass. Perioperative measures to reduce *S aureus* nares and skin colonization, administration of antibiotic prophylaxis, meticulous wound closure/care, and therapy directed to optimize patient host defense regulation mechanisms (eg, temperature, oxygenation, blood sugar) can minimize SSI occurrence. Antibiotic therapy for aortic graft infection should utilize bactericidal drugs that penetrate bacteria biofilms and can be delivered to the surgical site both parenterally and locally in the form of antibiotic-impregnated beads or prosthetic grafts.
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SURGICAL SITE INFECTION (SSI) after aortic intervention follows a predictable series of events resulting in bacterial colonization of the wound and graft. The implanted aortic graft or endovascular stent-graft is most susceptible to colonization during the early (<1 month) postoperative period, either from bacteremia or, more commonly, the adherence of pathogenic strains to the device with the development of a bacterial biofilm—an organism-produced microenvironment protective from host defenses and antibiotics. Analysis of patient risk factors and the type of aortic intervention reveals a higher observed SSI incidence of 5% to 15% compared to the <3% rate predicted by Centers for Disease Control and Prevention's National Nosocomial Infections Surveillance System for "clean" procedures in risk index categories 1 and 2.[1-4] Occurrence of SSI is higher after prosthetic grafting because the foreign biomaterial provides a more suitable microenvironment for bacterial adhesion and biofilm formation. Surgical site trauma with tissue injury, lymphatic disruption, and hematoma formation are common sequelae of graft implantation; especially if an incision to expose the common femoral artery was performed. Skin and subcutaneous tissue trauma can impede wound healing and provide a biologic substrate for bacteria biofilm formation, even in the absence of a prosthetic graft. Bacteria contact the surgical site by several mechanisms, including colonized mural thrombus of a diseased atherosclerotic plaque or aneurysm, bacteremia, bacteria transport to the wound via lymphatic channels, and patient contamination of the surgical incision by nose-to-hand transmission of bacteria. A surgical incision with wound drainage is more susceptible to surface bacteria biofilm formation and, if not addressed promptly, can result in SSI, with its attendant increased morbidity and healthcare costs. The majority of infections develop during the early postoperative period. The result is prolongation of hospitalization, further antibiotic therapy, and additional surgical procedures for wound debridement and closure procedures, followed by home healthcare and outpatient clinic visits to monitor surgical site healing. Late (>1 month) development of aortic graft infection is most commonly the result of *Staphylococcus epidermidis* colonization of the pros-

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Table 1 Classification of Aortic Graft Infection and Recommendation Regarding in situ Reconstruction

Intra-Cavitary Graft Involvement	In situ Revascularization Option
Graft-enteric fistula-associated mycotic aneurysm	Not recommended
GEE (graft erosion into gut)	FPV grafting
Total graft involvement with infection	
Biofilm infection (*Staphylococcus epidermidis*)	FPV or rifampin-impregnated graft
Virulent bacterial sp	FPV grafting
Aortofemoral graft limb Infection (localized to groin segment)	
Invasive infection	FPV grafting
Biofilm infection (*S epidermidis*)	Rifampin-impregnated graft
Aortofemoral graft limb infected due to contiguous infection	Not recommended
Diverticulitis	
Appendicitis	

Abbreviation: FPV, femoris profunda vein.

thesis producing a low-grade indolent graft surface biofilm infection.[5,6]

Aortic graft infection can be localized or, conversely, involve the entire prosthesis. Classification, including suitability for in situ reconstruction, depends on the extent of graft involvement, virulence of the infecting organism, and whether graft-enteric erosion is present (Table 1). Infection involving the surgical site can be superficial, eg, cellulitis, deep incisional involving the subcutaneous tissue/fascia, or involving other areas than the incision itself, ie, organ/space, such as along the length of an implanted vascular prosthesis or as an intra-cavitary aortic graft infection. When a prosthetic graft or endovascular device is implanted, the incidence of SSI is calculated for 1 year, not only for the 30 days after the procedure.

Antibiotic therapy for established graft infection should always include parenteral, culture-specific drug therapy bactericidal to cultured or suspected organisms. Because the preferred bacteria mode of growth is as a biofilm, antibiotics capable of biofilm penetration and killing of slow-growing organisms are recommended. Local delivery of antibiotics to the surgical site in the form of antibiotic-impregnated beads or an antibiotic-impregnated vascular prosthesis has also been shown to be of value.[7,8] Temporary implantation of antibiotic beads adjacent to an infected graft can sterilize the surgical site and provide an environment suitable for either in situ replacement or graft-preservation therapy. Antimicrobials in pulsed wound irrigation solutions and use of antibiotic-impregnated grafts are other strategies that can be used in the treatment of aortic graft infection to reduce new biofilm formation on in situ replacement grafts or within the surgical site.

Epidemiology of Vascular SSI

Although virtually any micro-organism can produce an SSI or infect an aortic prosthesis, Gram-positive bacteria, especially *Staphylococcus aureus*, are the prevalent pathogens involved in approximately 75% of all cases.[2,5-8] As in other surgical disciplines, the microbiology of vascular SSI has changed, with an increased prevalence of antibiotic-resistant organisms, including staphylococcal strains. An audit of prosthetic arterial graft infections treated by our vascular surgery group demonstrated a fourfold increase in methicillin-resistant *S aureus* (MRSA) infection: from 10% in the 1990s to 40% since 2000. Arterial interventions requiring a groin incision were associated with a 12% incidence of SSI, the majority (75%) caused by Gram-positive bacteria. MRSA accounted for approximately one-quarter of vascular SSI, and two-thirds of the strains demonstrated vancomycin resistance (minimum inhibitory concentration >1). The trend of MRSA and other antibiotic-resistant bacteria strains producing vascular SSI has been reported from multiple vascular centers in the United States and Europe. A 2005 report from the University of Texas Galveston vascular group reported an SSI rate of 11% after lower-extremity bypass grafting, with *S aureus* involved in 64% of cases, half of which were caused by MRSA.[9] Today, MRSA should be suspected in any vascular patient presenting with an SSI, including patients with a nonhealing lower-limb amputation performed for ischemia. The changing microbiology of SSIs, especially the dramatic increase in MRSA infection, has implications for both screening for nares colonization before arterial intervention and appropriate antibiotic prophylaxis in vascular patients with multiple SSI risk factors. Patient outcomes are less favorable with a MRSA compared to methicillin-sensitive *S aureus* graft infection, with an increase in both 30-day mortality and morbidity.[10-12] Our vascular group has observed the use of antibiotic bead implantation, serial wound debridement, sartorius muscle coverage as part of in situ replacement, or graft preservation therapy is safe and effective, even in patients with MRSA aortofemoral or femorofemoral graft infection.[5-7]

Surgical site wound infections typically manifest clinically within 30 days with wound erythema, drainage, and tissue inflammation. The infectious process can by caused by Gram-positive bacteria, Gram-negative bacteria, or a mixed flora, and thus broad-spectrum antibiotic therapy should be administered. If the surgical site is draining fluid, cultures should be obtained and the patient hospitalized. The late presentation of vascular graft infection can be attributed to the bacteria biofilm nature of the infectious process and the low virulence of infecting bacteria, most commonly *S epidermidis*. But with time, a graft biofilm infection can evolve to a more virulent infectious process, with superinfection by other bacteria, such as methicillin-sensitive *S aureus* or MRSA, especially if graft cutaneous sinus tract develops. If the presentation of aortic graft infection includes Gram-negative bacteremia, graft enteric erosion should be suspected.

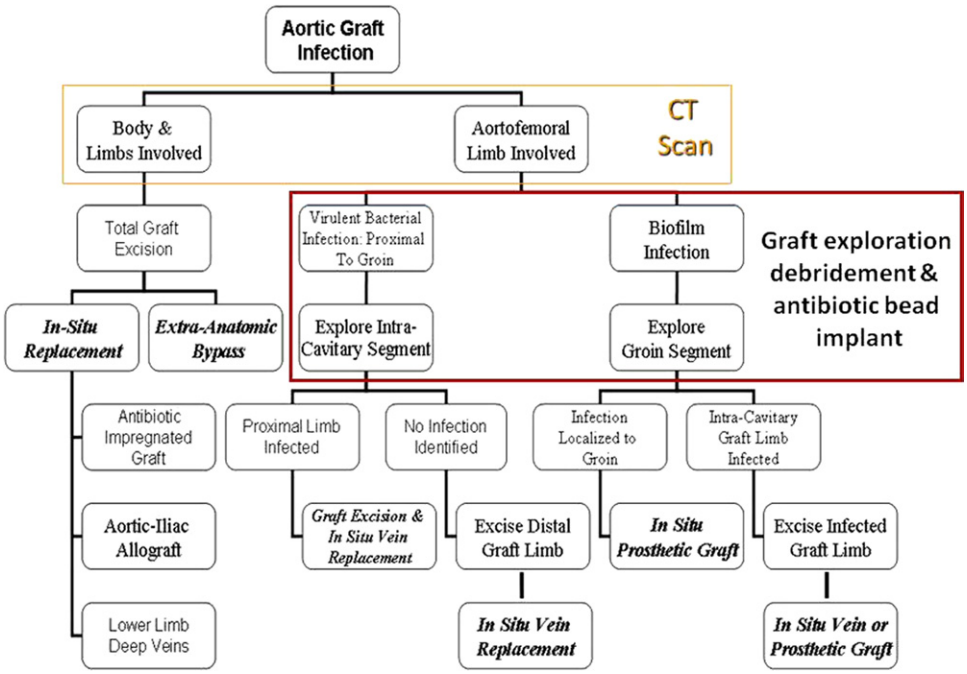

Figure 1 Algorithm for deciding on the type (vein *v* rifampin-soaked prosthetic graft) of in situ grafting procedure based on extent (entire *v* partial) of graft involvement and type (biofilm *v* virulent) of infection. CT, computerized tomography.

Overall, Gram-negative bacteria account for approximately 20% to 25% of vascular SSI, with the most common strains being *Escherichia coli*, *Pseudomonas aeruginosa*, *Proteus* sp, and *Klebsiella pneumoniae*. When confronted with a vascular SSI, initial antibiotic therapy should be guided by Gram stain of wound or perigraft fluid and, if Gram-positive organisms are identified, prompt antibiotic therapy with bactericidal killing properties to MRSA is recommended.[10,11]

Antibiotic Therapy

Broad-spectrum, bactericidal, parenteral antibiotic therapy should be started upon clinical suspicion of an aortic graft infection. The most common infecting organisms (in decreasing order of prevalence) are staphylococcal strains (*S aureus*, *S epidermidis*), streptococcus, *E coli*, *Kliebseilla*, Pseudomonas strains, and *Candida albicans*. MRSA now accounts for one-half of early and one-quarter of late aortofemoral graft infections. If *S aureus* or *S epidermidis* are the most likely pathogen, parenteral antibiotic therapy with a first- or second-generation cephalosporin and coverage for MRSA should be instituted. The preferred antibiotic for Gram-positive infection is daptomycin (6 mg/kg with dosing based on renal function) because of rapid, concentration-dependent bactericidal activity to all Gram-positive bacteria, including MRSA, and its ability to penetrate bacteria biofilms and kill bacteria in stationary-phase growth. Vancomycin and linezolid can also be used to treat MRSA infections, but these antibiotics have time-dependent bacteriostatic activity and do not penetrate bacteria biofilms; resulting in slower bacteria killing. In patients allergic to penicillin, administration of an aminoglycoside or a fluoroquinolone is recommended for extended Gram-negative bacteria coverage.[13]

Once the infecting organism has been isolated, eg, by needle aspiration of perigraft fluid or surgical exploration, antibiotic coverage should be modified based on antibiotic susceptibility testing of the recovered strains. Antibiotic therapy, including both parenteral administration and local delivery via antibiotic-impregnated beads and/or graft, is an adjunct to surgical management, which includes drainage of perigraft abscesses, debridement of infected tissues, excision of the infected graft, and restoration of lower-limb perfusion, whether by in situ replacement or extra-anatomic reconstruction.[7] Both are essential for a successful treatment strategy.

Surgical Therapy

Treatment Selection

The decision of whether a patient is suitable for in situ replacement depends on initial clinical presentation and the extent of graft infection and its microbiology as determined by operative exploration of the involved graft segment (Fig 1). The intent of the evaluation process is to accurately establish whether the infection involves the entire aortic graft or is localized to a graft segment; followed by a determination of whether a graft biofilm (amenable to in situ prosthetic grafting) or a more virulent infectious process exists. If surgical exploration demonstrates an invasive, virulent graft infection (organisms present on intraoperative Gram stain, positive perigraft tissue cultures), an autogenous vein in situ reconstruction should be considered. Preliminary implantation of

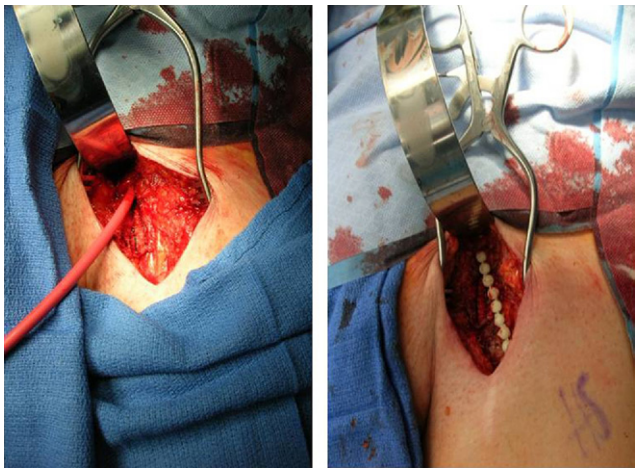

Figure 2 Implantation of antibiotic beads in groin before in situ graft replacement of an infected aortofemoral graft limb. (Left image) Red rubber catheter irrigation of perigraft cavity with "brown volcano," ie, hydrogen peroxide + povidone-iodine solution. (Right) Daptomycin beads placed in wound after cultures obtained; debridement and antibacterial irrigation completed. In situ graft replacement performed as a staged procedure 3 to 5 days later.

antibiotic-impregnated beads in the perigraft space is recommended to aid in surgical site sterilization. For Gram-positive infection, our group prefers implantation of daptomycin (1.5 g per 40 g bone cement powder) beads; for Gram-negative infections, tobramycin bone cement should be used. When possible, autogenous vein reconstruction of the excised graft segment should be performed in all cases, except when a localized biofilm infection is present. When an aortic graft infection is associated with graft-enteric fistula, extra-anatomic (axillofemoral) reconstruction in conjunction with graft excision and aortic stump closure should be considered.

In situ Reconstruction

If the infectious process is limited to an aortofemoral graft limb, a combined inguinal and lower abdominal oblique ("transplant") retroperitoneal incision can be used for surgical exposure. In the presence of extensive inguinal inflammation or abscess, a staged operative approach is recommended. At the initial operation, the perigraft abscess is drained, necrotic tissue excised, and the cavity irrigated with the so-called *brown volcano* solution composed of one-half strength hydrogen peroxide containing 10 mL povidone-iodine per 500 mL (Fig 2). This solution is used to cleanse the surgical site. Antibiotic beads fabricated within a plastic bead mold are then placed adjacent to the infected graft and the wound is closed (Fig 2). The second stage is performed 3 to 5 days later after culture results are available.[8] Exploration of the proximal aortofemoral graft limb is performed to assess for the presence of graft incorporation. If found to uninvolved with infection, ie, no perigraft fluid, graft incorporated with surrounding tissue, the proximal graft limb is clamped and transected. The distal graft is then excised to the femoral anastomosis and the graft bed is irrigated with antibacterial solution (clorpactin, 3 g/L) using a pulsed wound irrigation system. Depending on the earlier culture results, either deep vein or a rifampin-bonded polytetrafluoroethylene (6 or 7 mm diameter, Gel-Seal; Vascular Ltd, Glasgow, UK) is implanted.

Before wound closure, all surgical fields are pulse-lavaged again with the chlorpactin solution, and if a rifampin graft is used, the external graft surface is resoaked with the rifampin (60 mg/mL) solution. If Gram stain indicates a Gram-negative infection or graft-enteric erosion is present, tobramycin powder is spread along the arterial reconstruction and at the anastomotic sites. Perigraft fluid cavities and empty graft tunnels are drained using flat, closed-suction drain systems. Closed-suction drains are also placed in the beds of the excised femoral vein and the sartorius muscle, if used to cover the extracavity segment of the in situ graft reconstruction. On occasion, we have left antibiotic beads in the superficial portion of the groin wound for 7 to 10 days. The beads are attached to a 2-0 polypropylene suture brought through the skin and attached to a button for extraction at the bedside.

Parenteral antibiotic therapy administration is modified based on the explanted graft cultures with the intent to maintain bactericidal serum levels for 4 to 6 weeks after the in situ grafting procedure. Before discharge from the hospital, a baseline computerized tomography scan of the abdomen and femoral regions is obtained. This scan is repeated at 3 months and then every 6 to 12 months, depending on the type of in situ reconstruction. In general, prolonged oral antibiotic therapy is not prescribed after femoris profunda vein reconstruction, but is used for at least 3 months after in situ prosthetic reconstruction. The oral antibiotic prescribed is selected based on graft culture results and antibiotic susceptibility testing.

Risk Factors for Vascular SSI

The majority of arterial surgery procedures are classified as "clean" Class I by the National Research Council because the operative exposure and revascularization is performed in uninfected tissues without inflammation; the respiratory, alimentary, or infected urinary tract is not entered; and the wound is closed primarily with suction drainage if necessary.[1] Although diseased arteries can harbor bacteria, most commonly *S epidermidis* strains, within atherosclerotic plaque or mural thrombus, the inoculum and virulence are considered low. This observation is an important rationale for routine antibiotic prophylaxis. Arterial revascularization is not recommended in patients with invasive remote infection or bacteremia, except when the intervention is judged to be life-saving or for treatment of a mycotic aneurysm. In patients with critical limb ischemia and a clinical presentation of foot sepsis or wet gangrene, initial management should be surgical debridement of infected tissues in conjunction with antibiotic therapy; followed by open or endovascular revascularization when the invasive infectious process has been controlled.

Development of an SSI involves a complex interaction between a bacteria inoculum, host defense mechanisms, and surgical site healing. Audits performed in US hospitals

Table 2 Patient, Procedure, and Environmental Risk Factors for Surgical Site Infection

Patient-related risk factors
Nasal carriage of *Staphylococcus aureus*
Prolonged preoperative length of stay
Postoperative bacteremia
End-stage renal disease
Obesity
Malnutrition/low serum albumin
Older age
Smoking/nicotine use
Diabetes mellitus
Prior incision site irradiation
Malnutrition/low serum albumin
Autoimmune disease/corticosteroid therapy
Malignancy/chemotherapy
Procedure-related risk factors
Femoral/groin incision
Remote infection
Biomaterial implant
Emergency/reoperative procedure
American Society of Anesthesiologists score >2
Extended operative time
Hypothermia
Shock
Hyperglycemia
Environmental risk factors
Operating suite ventilation—environmental surface cleaning
Instrument and vascular implant sterility
Surgical attire and sterile operative technique

of SSIs have identified patient-, procedure-, and environmental-related risk factors that increase the risk for postoperative infection (Table 2). For the vascular patient, the most significant risk factors are: nasal colonization with methicillin-sensitive *S aureus*/MRSA, presence of a groin incision, prosthetic grafting or patch angioplasty, lower-limb arterial bypass grafting, postoperative bacteremia, and end-stage renal disease. Characteristics such as obesity, advanced age, smoking, and diabetes, all shown to be risk factors for SSI, are also present in many vascular patients.

Approximately 5% to 15% of healthy persons persistently carry *S aureus* in their nares, with the prevalence increasing in patients with end-stage renal disease (30% to 45%); active skin infection (15% to 20%); immune deficiency states, such as infection with human immunodeficiency virus (>50%); or residence in a long-term care facility (>50%). Preoperative *S aureus* carriage has been shown to increase the risk of SSI by four- to eightfold in patients undergoing cardiothoracic, neurosurgical, and orthopedic procedures. The cause-and-effect mechanism is believed to be patient transmission of bacteria from the nares to the surgical site(s) via their hands. In vascular patients, MRSA colonization has been shown to increase frequency of any nosocomial (wound, blood, urine, lung) infection (44% incidence and odds ratio of 4.5) and, in patients who develop a MRSA infection, hospital stay was prolonged by a mean of 4 days.[14,15] Randomized clinical trials of decolonizing therapy with application of intranasal antibiotic ointment (mupirocin calcium) before and after surgical procedures have been conducted, but have shown minimal to no reduction in SSI. Mupirocin, a topical antistaphylococcal agent that inhibits RNA and protein synthesis, eliminated *S aureus* carriage in 83% of colonized patients (23% of study population) compared to no effect in the placebo group. The odds of an *S aureus* carrier developing SSI was 4.5 times that of a noncarrier ($P < .001$), and although there was a trend to reduced *S aureus* SSIs (38% reduction), the differences between the treatment and placebo groups were not significant (7.9% *v* 8.5%).[12] Of note, prolonged use of topical mupirocin has been associated with the development of resistant strains. An audit of patients admitted to our vascular service for elective arterial intervention demonstrated an overall 6% incidence of MRSA nasal colonization, but higher (35%) in the end-stage renal disease patient population. Risk factors for MRSA colonization/infection mirror those of nasal *S aureus* colonization (Table 3). A recent report from Switzerland indicated a policy of universal screening of surgical patients for MRSA before admission, and nares decolonization in carriers may not decrease nosocomial infection.[14] To produce a significant decrease in SSI infection, specific surgical site care and antibiotic prophylaxis care directed at antibiotic-resistant bacteria are necessary.

Procedure-specific risk factors for aortic graft SSI include "open" versus endovascular intervention, presence of a femoral groin incision, and prosthetic graft/patch usage. If a procedure lasts >3 hours, produces shock or hypothermia, or requires blood transfusion, the likelihood of SSI increases. Intraoperative hypothermia of 1°C to 1.5°C increases the relative risk of postoperative infection twofold. Graft infection is rare after stent-graft abdominal aortic aneurysm repair (<0.5%), uncommon after intracavitary aortic graft implant (<2%), but in the range of 5% for aortofemoral graft reconstruction for aneurysm or oc-

Table 3 Risk Factors for Methicillin-Resistant *Staphylococcus aureus* Infection

Known previous MRSA infection
Immunosuppression
Diabetes mellitus type 1
Chronic open wounds
Previous antibiotic use within 90 days
Central venous catheterization
Residence in a long-term facility
Prolonged hospitalization
ICU admission
Dialysis
Advanced age
Hospitalization before onset of infection

Abbreviations: ICU, intensive care unit; MRSA, methicillin-resistant *Staphylococcus aureus*.

clusive disease. The increased SSI rate of procedures with a groin incision is related to tissue injury associated with the procedure, genital skin colonization, edema produce by lymphatic disruption, and failure of the skin edges to promptly heal in the groin region. The development of wound hematoma or incision separation caused by dermal or underlying fat necrosis reduces the bacteria inoculum required to produce an invasive infection. These wound problems occur more frequently in the groin, especially in the clinical setting of a redo arterial construction or an obese, diabetic patient. Any condition that impairs primary wound healing increases the likelihood of an SSI. This includes failure of prosthetic graft healing by incorporation of surrounding soft tissue. The finding by duplex scanning of perigraft fluid after polytetrafluoroethylene or polyester arterial bypass grafting beyond 3 to 4 months after operation is abnormal and suspicious for a low-grade graft infection, especially if associated with local signs of inflammation.

Preventive Measures

A multipronged approach is required to minimize the occurrence of vascular SSI, including attention to the pre-, intra-, and postoperative preventive measures published by the Centers for Disease Control and Prevention in 1999.[13-16] These guidelines address aspects of patient preparation, sterile surgical technique, surgical team antisepsis, hand disinfection, incision care, and antimicrobial prophylaxis. Surveillance of patients for nasal carriage of *S aureus*, especially MRSA, as well as a review in each patient of the inventory of SSI risk factors, can identify the high-risk cases and prompt an individualized prevention strategy. The increasing incidence of drug-resistant Gram-positive infections after arterial surgery is a concern and serves to re-emphasize the importance of preventive strategies. Surgeons should recognize that expanding the coverage of antibiotic prophylaxis is not a primary solution. Instead, prevention strategies to decolonize the *S aureus* carrier in combination with meticulous wound care and thoughtful antibiotic prophylaxis is recommended. There is accumulating evidence that regulation of host defense factors, eg, body temperature, oxygenation, and blood sugar, are important in determining SSI risk in an individual patient. Care measures to maintain normal temperature during and after surgical procedures, use of insulin therapy to keep blood sugar levels $<$180 mg/dL, and pulse oximetry monitoring to ensure 100% hemoglobin saturation are associated with reductions in SSI rates. Supplemental oxygen in the immediate postoperative period improves incisional oxygen tension and decreases wound healing complications.[15]

Antimicrobial prophylaxis in vascular patients should include therapy directed at *S aureus* nasal colonization, parenteral antibiotic therapy to assure adequate tissue levels are achieved before the procedure is begun and throughout the procedure, and surgical site care to impede bacterial colonization of injured skin and soft tissue (Table 4).[11,12] For effective antibiotic prophylaxis, a first or second-generation cephalosporin alone or in conjunction with daptomycin should be administered 30 to 60 minutes before the procedure. Prophylaxis must be provided for both Gram-positive and Gram-negative bacteria. Use of daptomycin or vancomycin alone is not recommended. When vancomycin is used for prophylaxis, the drug should be administered 60 to 120 minutes before incision because drug distribution to tissues and bacteriocidal activity is achieved more slowly. Cephalosporin antibiotics should be redosed if the procedure takes longer than 3 hours or if blood loss exceeds 1.5 L. If the patient is allergic to cephalosporins, aztreonam is an appropriate substitute. Antibiotic therapy is recommended for 24 hours.

Daptomycin is a concentration-dependent, bactericidal cyclic lipopeptide antibiotic with activity against all Gram-positive bacteria, including MRSA, penicillin-resistant streptococci, and vancomycin-resistant enterococci. The once-daily dosing, 2-minute intravenous infusion, rapid bactericidal activity within 30 minutes, and prolonged (18 to 24 hours) postantibiotic effect makes it particularly convenient and attractive for prophylaxis therapy. Daptomycin cannot be used alone for vascular procedure prophylaxis because it does not have activity for Gram-negative bacteria, which are known to cause one-quarter of vascular SSIs. A recent report from our vascular group of patients undergoing arterial reconstruction with groin incision demonstrated a reduction in SSI (5% *v* 18%; $P <$ .01) with the addition of daptomycin to a cephalosporin antibiotic prophylaxis regimen. In the MRSA-colonized patient requiring lower-limb prosthetic bypass graft, ie, a high SSI risk procedure, use of daptomycin prophylaxis for a total of 48 hours (two doses) is recommended.

A policy of universal screening for MRSA colonization should be considered in all vascular patients considered to be at risk for SSI based on patient- or procedure-specific characteristics.[12] A nasal swab of both nares should be submitted for polymerase chain reaction identification of bacteria. Results are typically available within several hours and testing is reimbursed by the Centers for Medicare and Medicaid Services. If screening is not possible, all patients should receive a prescription for mupirocin nasal ointment and chorhexidine antiseptic skin cleanser to use 3 days before an elective arterial revascularization procedure. Daily preoperative skin cleansing with chorhexidine produces a persistent antibacterial effect at the incision site after operation.

The importance of postoperative wound care cannot be overemphasized. Silver-coated wound dressings should be applied to groin incisions in the operating room and not disturbed for 24 to 48 hours unless wound drainage occurs. All dressing changes should be performed using sterile techniques with hand-washing before and after wound care is provided. If incision edges are traumatized, mupirocin ointment should be applied to produce an antibacterial barrier. If the surgical site demonstrates skin edge necrosis, hematoma, or profuse lymphatic drainage with surrounding tissue edema, a more aggressive wound management strategy is required. This might consist of operative exploration of the wound, antibiotic irrigation, closure suction drainage, and a secure skin closure technique.

Table 4 Treatment Strategies to Prevent Vascular Surgical Site Infection

Surveillance
Patient screening for nasal carriage of *Staphylococcus aureus* of MRSA
Decolonization
Preoperative intranasal mupirocin (applied to both nares for 3 days before and 2 days after operation)
4% chlorhexidine gluconate wipes or body washing at planned incision site to decolonize skin surfaces for 3 days before procedure.
Antibiotic prophylaxis
Low-risk SSI: carotid endarterectomy, percutaneous endovascular stent/stent-angioplasty
Cefazolin, weight-based, 1 to 3 g IV slowly 60 min before procedure, and repeated 1 to 2 g if procedure >3 h or blood loss >1.5 L. Dosing repeated every 8 h for 24 h; or cefuroxime 1.5 g IV 60 min before surgery and every 12 hours for total of 6 g. If patient has a cephalosporin allergy, give aztreonam 1 g IV 60 min before procedure and every 8 h for 24 h.
High risk: groin incision, prosthetic grafting, dialysis access procedures, lower-extremity bypass grafting, *Staphylococcus aureus* nasal carriage, history of MRSA infection, multiple risk factors
Add daptomycin 6 mg/kg (single dose) IV slowly 60 min before procedure; a second choice but not preferred prophylaxis, vancomycin 25 mg/kg IV (at 1 g/h) before surgical incision.
Antibiotic-impregnated prosthetic graft
Soak gelatin-coated polyester or PTFE vascular prosthesis in a rifampin (30 to 60 mg/mL) solution for 15 min
Antibiotic-impregnated PMMA (bone cement) beads
Daptomycin 1.5 g per packet (40 g) of PMMA, if Gram-negative activity required, add 2.4 g tobramycin powder
Postoperative care
Monitor serum blood sugar levels, with insulin therapy to keep < 180 mg/dL
Supplemental oxygen to keep pulse oximetry saturation >98%
Incision care
Femoral/groin incision: Apply silver-impregnated wound dressing (Acticoat) for 24 to 48 h; followed by topical mupirocin ointment to incision if wound drainage or injured skin edges present
Apply sterile dressing daily for 48 h
Wash hands before and after dressing changes
Educate patient and family about surgical site care and symptoms of surgical site infection

IV, intravenous; MRSA, methicillin-resistant *Staphylococcus aureus;* PMMA, polymethylmerthacrylate; PTFE, polytetrafluoroethylene; SSI, surgical site infection.

Summary

Antimicrobial-resistant pathogens, especially MRSA, are increasingly involved in aortic graft infection. Antibiotic therapy should include coverage for MRSA and should include patient assessment for nasal carriage for MRSA, which, if positive, should be treated using decolonization procedures and patient isolation to minimize transmission to other patients. The thoughtful use of antibiotic-based culture susceptibility, presence of a biofilm, and drug delivery both systemically and locally into the wound, are essential parts of in situ replacement or graft preservation therapy. In the MRSA-colonized patient, thoughtful antibiotic prophylaxis usage is required with a combination of daptomycin (5 mg/kg single dose) and cephalosporin (for Gram-negative coverage) is an effective strategy to reduce SSI. The entire surgical team must participate in institutional efforts to control nosocomial infections, antimicrobial resistance in bacteria, and SSI rates.

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